

Correspondence

The Editorial Board will be pleased to receive and consider for publication correspondence containing information of interest to physicians or commenting on issues of the day. Letters ordinarily should not exceed 600 words, and must be typewritten, double-spaced and submitted in duplicate (the original typescript and one copy). Authors will be given an opportunity to review any substantial editing or abridgment before publication.

Irish Spring Soap

TO THE EDITOR: I am writing regarding a possible new effect of Irish Spring soap. I have frequently seen patients with dry erythematous and pruritic skin from the use of this strong detergent soap. However, today I saw a patient with a prominent miliaral rash on the face, neck, upper arms, upper back and upper chest. By a thorough history we ruled out other possible inciting factors.

I think it is a certainty that this miliaral rash was caused by the Irish Spring and this should be added to the list of negative skin effects of this product.

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Primary Hypertension—A Disease Entity?

TO THE EDITOR: The article by Drs. Rudd and Morton in the September issue presented an excellent review of problems associated with treatment of hypertension [Nontraditional Problems of Anti-hypertensive Management]. However, the authors seem to take for granted that primary hypertension is a well-defined disease entity.

If such an entity requires such a complicated lengthy treatment plan, there must be more involved than just levels of blood pressure. Can they really define or classify this entity with pathologic correlation, varieties of natural course, etiologic possibilities and so forth, as in more documented diseases such as types of arteritis—Wegener granuloma, for instance?

In the 1966 edition of *Controversies in Internal Medicine*, Drs. Goldring and Chasis concluded (after 30 years' research) that treatment of hypertension was not successful. But they did not think

that treatment should be the real issue. They were concerned about the increasing emphasis in the literature on treating primary (then essential) hypertension with a corresponding decrease in studies on causes, pathologic findings and natural course; in other words, what is the real disease of which elevated blood pressure may be only a secondary or concomitant manifestation? In particular they were bothered by lack of knowledge about the relationship of hypertension to arterial disease, and disturbed that arterial disease progressed unabated in hypertensives whose blood pressure was reduced to normal by sympathectomy.

If we do not know any more about this now than in 1966 maybe, just maybe, primary hypertension is not really a disease entity. Perhaps there is a need to publish a few case reports.

JAMES W. KASCH, MD
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Of Decibels, 'Highs' and Privacy

TO THE EDITOR: Over in Iran, the Ayatollah Khomeini has banned all music, calling it the opiate of the masses. Here, in the land of the free and the home of inviolate human rights, we put this down as a despotic act of a senile dictator.

And yet, recalling the sights and sounds of the summer past, I have an uneasy feeling that the Ayatollah may be at least partly right. I remember that entranced faraway look in the eyes of the lad as he *tooled* slowly down the middle of our residential street, his radio tuned to top decibel. And I cannot forget how the young folks next door sent out throbbing, thumping, earsplitting beats through every open window to every unfortunate passerby.

Society is becoming aware of the physical dangers of noise, and is trying to abate noise pollution. In this lumbermill town, noise—loud

noise—is an accepted occupational hazard for the workmen. Noise dampening efforts are made, workers are encouraged to wear earplugs. But the saying is that when a millworker retires he is given not a watch but a gold-plated hearing aid. Out on our lake, motorboats are required to keep the noise of their exhausts below a certain level and underwater mufflers are mandatory.

But so far no legislator has dared suggest that mufflers be put on radios or decibel governors on stereos, even though the hearing acuity of future generations is in jeopardy. My audiologist friend suggests, half seriously, that by the year 2000 infants will be born with a hearing aid in place.

Everyone agrees we have a problem but we lack consensus on a solution. A first step might be to search for a cause of this phenomenon. A psychiatrist at the University of Chicago suggests our narcissistic younger generation turns up the volume to attract attention, the "Hey, look me over" syndrome. Another research worker maintains that the monotonous, repetitious sounds allow youths to block out the world of reality, while achieving their personal *high*. A less scholarly observation, undoubtedly made by a parent of a teenager, is that this hard-rock racket is simply an angry blast at the Establishment.

Whatever the explanation, the fact remains that these loud and pulsating sounds are an invasion of a person's privacy, as well as a cacophonous insult to his ear drums. Is there any hope for relief? The Ayatollah Khomeini's total prohibition would not work here. What we need is a forceful, grass-roots educational program.

Otolaryngologists of America, rise up in protest! Let your voices be heard. Mothers of America, make war on noise, lest universal deafness be the fate of future generations.

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Clonidine Withdrawal— Fact or Fiction?

TO THE EDITOR: The medical literature is unfortunately clouded by a maze of poorly defined terms to describe a syndrome occurring in some patients when antihypertension medication is abruptly stopped; these include discontinuation syndrome, acute posttreatment syndrome, acute withdrawal syndrome and rebound hypertension.

In a case report on clonidine withdrawal by Mate and his colleagues in the July issue (Mate TP, Swerdlin AHR, Stone RA, et al: Clonidine hydrochloride withdrawal complicating bilateral nephrectomy. *West J Med* 131:59-62, Jul 1979) several false and misleading statements are made concerning this syndrome. The authors attempt to incriminate clonidine withdrawal as the cause of "rebound hypertension." In the first place, the posttreatment blood pressures (250-270/100-120 mm of mercury) do not indicate any significant rebound over that of the pretreatment blood pressures (190-200/110-120 mm of mercury). Second, the authors state that "neither propranolol nor minoxidil withdrawal has been associated with a rebound phenomenon." The withdrawal syndrome is not unique to clonidine and has been reported to occur with bethanidine,¹ methyl-dopa²⁻⁵ and propranolol.^{6,7} Third, acute cessation of combination drugs especially a centrally acting antihypertensive in patients receiving propranolol may produce worse symptoms of blood pressure elevations than single agents alone due to uninhibited stimulation of vasoconstrictor alpha receptors during beta blockage.^{8,9} The authors provide evidence that extreme levels of catecholamines appear to be the sole pressor mechanism responsible for the sustained hypertension. However, other studies have shown no excessive rise in catecholamines upon acute discontinuation of clonidine.^{10,11}

It is interesting to note that abrupt cessation of clonidine (0.4 mg given orally twice a day) three days after its reinstitution in this patient did not result in another "rebound phenomenon." This is consistent with recent studies,^{11,12} suggesting that doses of clonidine less than 1.2 mg per day did not result in overshoot blood pressure or symptoms of sympathetic overactivity. However, it could just as easily be interpreted that the combination of propranolol and clonidine or propranolol alone was the cause of the withdrawal syndrome in this patient. This case only adds more confusion to a poorly defined syndrome. Interchangeable use of terms such as rebound hypertension and withdrawal syndrome should be avoided and an attempt made to distinguish between overshoot hypertension, return to pretreatment blood pressure, and sympathetic overactivity with or without hypertension. I do not believe it is justified in view of the above facts to implicate clonidine as the cause of this withdrawal syndrome. This case might just as easily